PROSPECTIVE STUDY OF EFFECTS OF TURP ON OUTCOME,MORBIDITY AND MORTALITY IN PATIENTS WITH NON DIALYSIS REQUIRING RENAL INSUFFICIENCY

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***AND* HOSPITAL**

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CERTIFICATE

This is to certify that the dissertation entitled **"PROSPECTIVE STUDY OF EFFECTS OF TURP ON OUTCOME, MORBIDITY, MORTALITY IN PATIENTS WITH NON DIALYSIS REQUIRING RENAL INSUFFICIENCY"** is the bonafide original work of **Dr.AMAR NEEDHI GANESAN.B** in partial fulfillment of the requirements for **MCH UROLOGY BRANCH – IV** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in August 2014. The period of postgraduate study and training was from August 2011 to July 2014.

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DECLARATION

I, **Dr. AMAR NEEDHI GANESAN.B ,** solemnly declare that the dissertation titled, "Prospective Study of effects of TURP on outcome,morbidity and mortality in patients with non dialysis requiring renal insufficiency" is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2011-2014 under the guidance of and supervision of **PROF.DR.V.SELVARAJ, M.S, M.CH URO,** Professor and Head, Department of Urology, Government Stanley Medical College, Chennai-600 001.

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ABBREVIATIONS

BPH - Benign prostatic hyperplasia

TURP – Transurethral resection of prostate

IPSS - International Prostate symptoms score

LUTS - Lower urinary tract symptoms

INTRODUCTION

Benign Prostate Hyperplasia (BPH) is a common disease in adult men and its incidence is age related. Prevalence of BPH is approximately 25% in men aged 40 to 49 years, 50% in men aged 50 to 59 years and 80% in men aged 70 to 79 years.[1]

Renal failure and symptomatic benign prostatic hyperplasia (BPH) are two common health problems, they usually co-exist in 5.9–13.6% of the male population over 50 years of age. Actually going by the natural history of the disease and its progression with relation to Benign prostatic hyperplasia and its complications, it is noted that 13.6% of patients who presented to undergo Transurethral resection were in renal failure. It is usually not clear in this group of patients whether the reason for renal insufficiency is or is not Benign prostatic hyperplasia. However, it has been reported in some studies that the incidence of diabetes mellitus and hypertension is higher in patients with renal failure (RF) and lower urinary tract symptoms (LUTS) due to Benign prostatic hyperplasia. On the other hand, it is known that due to chronic urinary obstruction, BPH can lead to renal failure and even death occasionally.

The main constant indication for BPH surgery has been medical treatment-refractory moderate or severe Lower urinary tract symptoms;

but definite surgical indications usually includes upper urinary tract deterioration. Renal failure increases the risk in transurethral prostatic surgery, so there is a tendency for avoiding the surgery till there is a detection of an absolute indication occurs. But these studies are based on data from two or three decades ago not representing current practice.

As we understand that these patients with BPH whether symptomatic or asymptomatic, if left untreated may present with renal failure which could be chronic or acute. Despite the many possible causes of renal failure in elderly patients, the common causes were BPH (38%), neurogenic bladder (19%), obstructive pyelonephritis (15%).[2]

While the underlying mechanism for developing renal failure associated with benign prostatic hyperplasia is likely multifactorial and co-morbid factors in elderly men may contribute to renal impairment, we wanted to evaluate the incidence of BPH with renal failure at our institute.

TURP remains the gold standard surgical procedure for treatment of these cases. However, patients in renal failure have an increased risk for complications after TURP compared with patients with normal renal function, so we wanted to study the treatment outcome and complications associated with its management.

Attending to high prevalence of BPH in older men with CKD it is invaluable to take into consideration the relationship between these two clinical entities. However, despite the high prevalence of renal failure and BPH in elderly men, there is limited knowledge on the association between these two conditions, there is very little information in the literature regarding the role of only BPH as a causative factor in causing renal failure and its treatment outcome.

The purpose of this study was to determine the incidence of renal failure associated with BPH , effect of TURP in the morbilidity and mortality of patients with renal failure.

AIMS AND OBJECTIVES

- To study the prevalence of co-morbid factors in patients with Benign prostatic hyperplasia and non dialysis requiring renal insufficiency.
- Study of treatment outcome following the surgical management of BPH with non dialysis requiring renal failure.
- Study of complications associated with operative management in these patients.

REVIEW OF LITERATURE

Epidemiology of Benign prostatic hyperplasia :

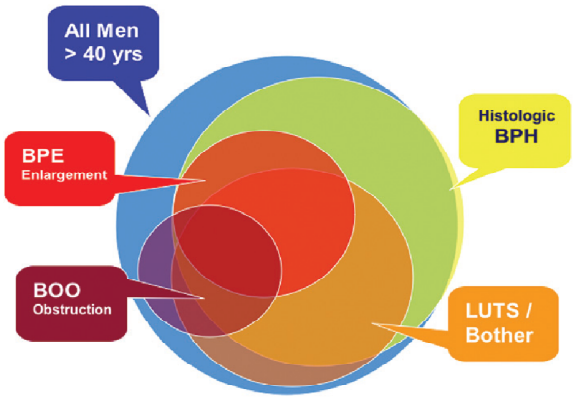
Benign hyperplasia of Prostate occurs with increased growth of non malignant tissue of prostate which surrounds urethra, it narrows the lumen of urethra and subsequently gives rise to symptoms.[63]

Diagnosis of benign prostatic hyperplasia done conclusively on histological evaluation of prostate. Histological evaluation is by taking prostate by transurethral resection or trans rectal ultrasound guided or by doing autopsy. But other measures, namely symptomatology, obstruction of bladder with associated enlarged prostate is used to mark benign prostatic hyperplasia. Because of this, the end point of benign prostatic hyperplasia becomes difficult to assess.

These facts gives us sufficient details about the newly occurring cases and disease progression.The prevalence of benign prostatic enlargement is calculated by taking histological part (assessed by autopsy) or clinically. [(3)] No men 30yrs and younger had evidence of benign prostatic hyperplasia and the peak of prevalence increased along with each age group, ultimately reaching 88% in men with eighties.[4]

Three things which are assessed separately which comprise of symptoms[3]; enlarged prostate with obstruction.[5] It can present as all the

three or two of them or only one. Hence prostatism has been now changed to lower urinary tract symptoms (LUTS).[5,6]



Representative diagram showing relationship of various factors involved

Histologically, hyperplasia of prostate is seen in 8% men of age 31-40. Whereas it increases to 90% of men in ninth decade.[4,7]. In the setting of Benign prostatic enlargement, untreated chronic kidney disease can result in ESRD requiring dialysis or kidney transplantation.

Relationship between benign prostatic hyperplasia and Chronic Kidney Disease :

Etiology of benign hyperplasia of prostate is largely unknown, but from clinical practice and studies, natural history of hyperplasia of prosate leads to urinary obstruction, landing in deterioration of renal

function with time. Both benign hyperplasia of prostate and chronic kidney disease are commonly prevalent in ageing male.

Following are the studies conducted to find relationship between Benign hyperplasia of prostate and chronic disease of kidney:

a. Study conducted by Epidemiology Project of Rochester, concluded that association was significant between signs & symptoms of benign prostatic hyperplasia and Chronic kidney disease in their population-based sample which comprised of white men numbering 476.[9]
b. But, another study conducted in Austria concluded that Lower urinary tract symptoms in men was not an independent risk factor for impaired kidney function.[10]
c. Another study that was conducted on 30,466 men study from the HUNT II (the Nord-Trondeleg Health Study), they also failed to show a connection between Lower urinary tract symptoms and Chronic kidney disease.[11]
d. But recent evidence from two different studies have found out an association of bening hyperplasia of prostate and chronic disease of kidney.[12,13]

Eventhough the prevalence of chronic kidney disease is considered to be low in men with benign hyperplasia of prostate, we should stongly

consider it in patients presenting with obstructive lower urinary tract symptoms mainly or with low peak flow rate or patients having hypertension or diabetes. [13]

Clinical guidelines for benign hyperplasia of prostate, which was created in 1994 by Health Care Policy and Research Agency in 1994, recommended routine serum creatinine measurement in men with lower urinary tract symptoms, but in year 2003, a update discontinued the routine serum creatinine measurement in these persons.[9] So these different approaches to benign prostatic hyperplasia patients, leads to a significant amount of patients with Chronic kidney disease going undetected.

By taking this data into account, we should always bear in our minds that benign prostatic hyperplasia is almost an ubiquitous condition in the setting of old men. Low occurrence of chronic kidney disease in benign prostatic hyperplasia usually in clinical trials, should not be used to infer a weak association between the two disease processes.

Signs and symptoms of Bladder outlet obstruction namely low QMax, high residual urine (post void), Obstructive symptoms of lower urinary tract are significant predictors of chronic kidney disease.[9,12] Bladder outlet obstruction, probably, is the bridge connecting between between chronic disease of kidney and benign hyperplasia of prostate.[13]

But we should never forget that chronic disease of kidney in the setting of benign hyperplasia of prostate is a multifactorial process, and thus it becomes difficult to separate the contribution of this condition from all the other renal insults. This also affects and it takes its toll on the design of studies as many men with concomitant disease are excluded, and thus makes it harder for investigators, to take into account the true influence of both these conditions.

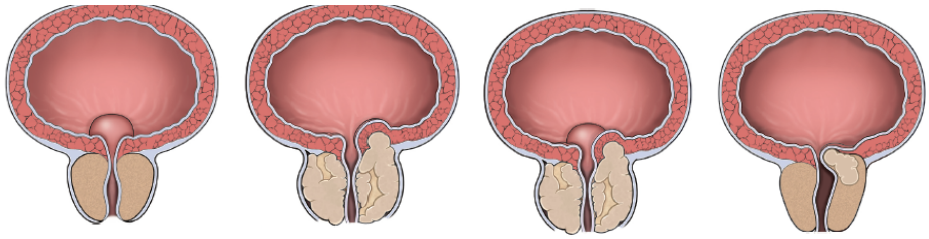
Pathophysiology and Progression of Benign prostatic hyperplasia :

Although the etiology of benign hyperplasia of prostate is not known, there are similarities found between benign hyperplasia and morphogenesis of prostate during development. This prompted to suggest hypothesis that benign hyperplasia of prosate results due to reawakening of embryogenesis in adulthood.[14]

Most common renal pathology finding in men with obstructive nephropathy with benign prostatic hyperplasia is, Chronic interstitial nephritis[15, 9] and 30% of cases have been attributed to obstructive uropathy. End stage disease of kidney secondary to bladder outlet obstruction can be prevented if recognised early, however it is still difficult to recognise which men with benign prostatic hyperplasia can develop renal failure.

So we have to recognize factors which are measurable during evaluation of benign hyperplasia of prosate. This helps to prevent patients from developing renal failure.

Anatomical



Benign hyperplasia of prostate initially in periurethral transition zone, then progressively enlarging

Benign hyperplasia of prosate develops first in periurethral *transition zone* of the prostate. This transition zone has two separate glands located external to the preprostatic sphincter. Benign prostatic hyperplasia also involves an increase in the number of glands, mostly the periuretheral glands increase in number, simultaneously there is a increase in smooth muscle and connective tissue in the periuretheral region of prostate.[16,3]

Physiological

According to the physiological point, when the prostate enlarges, urethral compression occurs, which prevents the outflow of urine and contributing to the common lower urinary tract symptoms. Smooth muscle cells usually are not optimal for force generation. Non muscle

myosin heavy chain is upregulated along with down regulation of smooth muscle myosin heavy chain. These things suggest that there is loss of normal modulation pathways.[16]

We need to elucidate the factors determining passive tone in prostate.[(3)]Adrenergic system regulates the active muscle tone in prostate.[(18)] Adrenergic neurotransmitters have been involved in prostate smooth muscle regulation as well as contraction, and α-adrenergic blockade actually leads to a significant normal protein gene expression downregulation, specifically smooth muscle myosin heavy chain.[19,3]

Clinical

Studies conducted recently, found there was a significant correlation between urgency symptoms and prostate size.[20] This also proves a link between Chronic disease of kidney and benign hyperplasia of prostate. Prostate size assessed by rectal examination is considered to have poor reliability, so trans rectal ultrasound of prostate is used in studies.[3]

So, prostate and its benign enlargement can contribute for outflow obstruction, by two factors, one is the static component (periurethral compression caused by stromal component), the second one is also by dynamic component (smooth muscle cells and supplying adrenergic pathway).

BENIGN PROSTATIC HYPERPLASIA

PATHOPHYSIOLOGY AND PROGRESSION :

Disease progression :

Three things contribute, they are Symtoms of lower urinary tract, Chronic retention of urine, Acute retention of urine.

Lower Urinary Tract Symptoms (LUTS)

Lower urinary tract symptoms (LUTS) are described as clinical criteria to define a man with urinary problems. Most men with benign hyperplasia of prostate have following symptoms namely voiding dysfunction, nocturia, urgency, thin urinary stream, increased urinary frequency and a sense of incomplete bladder emptying.

Many studies were done to achieve a scientific relation between Lower urinary tract symptoms and Chronic kidney disease. But, until recent years there was no palpable evidence mainly connecting these two entities. A retrospective study which was done to find this, did not find any relation between duration of symptoms and serum creatinine levels measured in these patients.[21]

Our clinical practice shows us that many men with Lower urinary tract symptoms routinely do not value their symptoms, and they avoid seeking medical care. Those older men often tolerate their symptoms and disregard their lower urinary tract symptoms avoiding consultation.[22]

However we must take into account that the absence of these symptoms in older men does not necessarily exclude benign prostatic hyperplasia with urinary outlet obstruction. Silent prostatism or Silent obstruction, a term which describes asymptomatic patients developing chronic disease of kidney resulting from benign hyperplasia of prostate. So, International prosate symptom score will not be useful in these patients.[23]

Post-voiding residual urine volume – Chronic urinary retention

Chronic urinary retention is the mechanism, by which benign prostatic hyperplasia can result in renal failure.[9] Chronic retention of urine is taken as post-void residual urine (PVR), higher than 100 mL. It is significantly associated in Chronic disease of kidney.[24,9,12]

Acute urinary retention

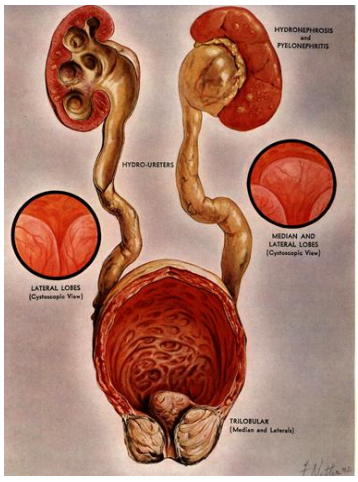
Acute urinary retention is described as an acute complication of Benign prostatic hyperplasia. These patients suffer from an acute, sudden and painful inability to void. It needs emergency intervention. Acute urinary retention was the main indication of surgery in 25% and 30% of men undergoing trans urethral resection.[3] Other causes that can trigger acute urinary retention are surgery, anaesthesia, trauma, medications, medical examination and urinary tract infections (mainly prostatitis).

Acute urinary retention is uncommon in men under sixty years, and may be responsible for the majority of acute renal failure cases due to obstructive uropathy.[25] Men in whom acute urinary retention is promptly relieved by catheterizing the bladder, acute renal failure usually does not develop but long-term tubular dysfunction can still occur.[9]

So, dysfunction of renal tubules can persist after the acute urinary retention episode and probably this might result in progressive renal disease affecting these patients.

Pathophysiology of Progression to Renal failure

Remodelling of Bladder in response to urinary obstruction



Enlarged lateral lobes and median lobe of prostate producing bladder outlet obstruction with bladder Trabeculations which leads to bilateral hydrouretreronephosis

The bladder plays an important role in pathophysiology of benign hyperplasia of prosate and its complications. So, the bladder responds by adaptation, though it is a partial adaptation. Obstruction induces changes in bladder is closely related to symptoms rather than the obstruction by itself.

Two types of bladder changes are observed. First is detrusor instability presenting with frequency and urgency and second one is decreased contraction of detrusor leading to poor stream, hesitancy, intermittency, increased residual urine. [3]

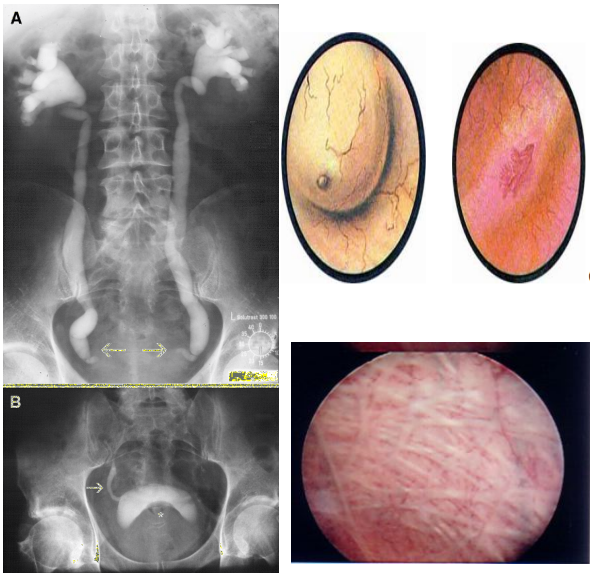
Thickened bladder is measured by ultrasound. Trabeculations due to hypertrophy of smooth muscle and permeation of connective tissue accounts for elevated bladder pressure in patients with high pressure chronic retention.[26]

The major detrusor changes and trabeculation were due to an increase in detrusor collagen. Severe trabeculation is directly related to significant post void residual urine[27] suggesting that increases in collagen in the bladder wall is mostly responsible for incomplete bladder emptying rather than impaired detrusor muscle function.[3]

Detrusor hypertrophy is one of the first and foremost modification in the bladder and, as studied in animal models, the response initially is the hypertrophy of smooth muscle.[27]

Cellular and physiological changes that occur in bladder muscle and collagen, contributes to high pressure bladder that perpetuates itself with worsening ability to empty and causing kidney lesions. These mechanisms of bladder remodelling develop in a hypofunctional bladder, with low compliance.[28]

Ureterovesical junction and upper tract dilation



Intravesical enlargement of prostate producing bilateral ureteric dilatation with ureterovesical junction obstruction and bladder trabeculations

Generally, ureterovesical junction obstruction caused by remodelling of bladder in chronic urinary retention is a contributing mechanism for renal failure in benign prostatic enlargement.[9] Upper tract dilation occurs as a consequence of a continuing bladder outlet obstruction and remodelling (detrusor hypertrophy and scarring) which leads to anatomical ureterovesical junction obstruction.[29]

Upper urinary tract dilation or hydronephrosis is consistent with chronic renal failure from obstructive uropathy. In men with benign prostatic hyperplasia and increased serum creatinine, hydronephrosis is common in about one third of patients, but it is found in 90% of men with benign prostatic hyperplasia who are hospitalized with uremic symptomatology. In ultrasound evaluation, it is common among patients with bilateral hydroureteronephrosis, leading on to compression and renal cortical thinning, with obvious impact in renal function.[23]

Other causes

- Recurrent urinary tract infections with chronic retention due to benign hyperplasia of prostate may be a contributing factor leading on to chronic renal failure.[9]
- Other cause being, Secondary hypertension occurring due to chronic urinary retention is a described complication of benign prostatic hyperplasia, leading to hypertensive kidney disease.[30]
- Nephrogenic diabetes insipidus can be caused by partial or chronic urinary obstruction, this can result in renal failure.[31]

Other clinical entities like diabetes and hypertension are independent factors that can lead to chronic kidney disease.[(32)]

Patients with benign prostatic hyperplasia are usually carriers of these pathologies above mentioned that are likely to seriously aggravate

renal function and must be taken into account as sombre conditioners of renal disease.

Clinical symptomatology :

Benign prostatic hyperplasia is a chronic and progressive condition,[33] where in patients generally have a history of lower urinary tract symptoms and indolent obstructive uropathy. The clinical presentation of benign hyperplasia of prostate and associated obstructive uropathy varies and this reflects the source and duration of obstruction. In benign prostatic hyperplasia, symptoms directly results from bladder outlet obstruction (BOO) from prostate enlargement which is static component dynamic component by enhanced smooth muscle tone and increased resistance within the gland.

These physiologic issues reflect in voiding dysfunctions, that significantly affects the health and quality of life of older men. Most of the patients have characteristic symptomatology.

Patients complaints are usually thin urinary stream, nocturia, urgency, with decreased flow rate, low values in Qmax and Qaverage, a sense of incomplete bladder emptying, straining during micturition, increased urinary frequency and dribbling during or after urination.[9]

Physical examination consists of digital rectal examination (assesses the prostate characteristics and volume), bladder distension by

lower abdominal percussion and palpation and focused neurological examination to rule out neurogenic component involved in the presentation.

Recurrent or persistent urinary tract infections (UTI) which are associated with prolonged urinary stasis of lower urinary tract obstruction, urgency, frequency, dysuria, hematuria are common complaints among men with Urinary tract infection.

Chronic urinary retention due to benign hyperplasia of prostate is defined as a palpable bladder, corresponding to high post void residual urine[34] and most of these patients with chronic urinary retention have an indolent and progressive disease, with worsening of urinary symptoms and the majority of these patients just seek for medical care in worsening health conditions with acute renal insufficiency.

It becomes necessary to investigate the symptoms and signs suggesting chronic kidney disease like vomiting, nausea, edema, lethargy,hypertension.[23]

In some rare cases of patients who present to the emergency room because of anuria, require interventional procedures like indwelling catheter, nephrostomy tube insertion either unilaterally or bilaterally and sometimes depending on the level of renal function, hemodialysis may be required.

Even though the signs and symptoms of benign hyperplasia of prostate are normally present in a group of patients, there are a significant number of patients who are relatively asymptomatic[35] (without significant voiding dysfunction), but can present primarily as a clinical sequel of renal insufficiency namely uremic symptoms; with vomiting, nausea, mental status changes and electrolyte disturbances namely hypercaliemia and nonanion gap acidosis.

Older patients with voiding dysfunctions caused by chronic urinary obstruction might present with hypertension due to hypervolemia in the case of bilateral obstruction or increased renin release.[35] Hypertension, on other hand can be itself the sole cause of renal failure.

If left untreated, benign prostatic hyperplasia can cause serious complications including hematuria, urinary tract infections, renal failure, bladder stones, incontinence and mortality related with benign prostatic hyperplasia.

Complications in Patients with Benign hyperplasia of prostate :

Mortality :

Patients with renal failure undergoing transurethral resection have a higher risk (25%), in comparison to normal renal parameters (17%), whereas mortality increases sixfold.

Bladder stones

Bladder stones occur in line with retention of urine leading on to stasis and urinary infection. These factors favour ion aggregation and stone nucleation. This leads on to formation of vesical calculi.

Urinary tract infections

Urinary tract infections are usually due to chronic urinary obstruction, due to increased post void residual urine, predisposing to Urinary tract infections.[39] This leads to multiple hospital admissions for treating these urinary tract infections.

Incontinence of Urine

Incontinence can develop from transurethral surgical intervention for benign prostatic enlargement [40]. Other causes are overflow incontinece due to overdistention of the bladder or due to instabilityof detrusor resulting in urge incontinence.[41,42,3]

Hematuria

Gross hematuria with clots can be seen in patients with benign hyperplasia of prostate. Microvessel density in prostate is higher in hematuria patients than in controls[3] suggesting that vascular lesions in the prostate.

Tests for diagnosing the condition :

Nowadays it is increasingly rare to find a patient with chronic renal failure from chronic urinary retention due to benign prostatic hyperplasia, about 13.6% (range from 0.3 to 30%) of men with benign prostatic hyperplasia may present with chronic kidney disease defined by a baseline serum creatinine of more than 133 mmol/L (1.5 mg/dL). This is particularly true in older patients with cognitive deterioration and autonomy impairment.

In order to diagnose and monitor the impact of a bladder outlet obstruction due to benign prostatic hyperplasia in the upper urinary tract, some laboratory and imaging tests:

- Standardized questionnaires
- Creatinine levels in serum or measuring estimated glomerular filtration rate (eGFR)
- Urine analysis
- Serum prostatic specific antigen (PSA) levels
- Uroflowmetry with peak flow rate determination (Q max)
- Renal and bladder ultrasonography with detrusor thickness evaluation
- Transrectal prostate ultrasonography with measurement of prevoid urine and post-void residual urinary volume

- Cystometry, other urodynamic studies as needed
- Urethrocystoscopy.

Symptom assessment by standardized questionnaires

Benign prostatic hyperplasia Impact Index (BII), a questionnaire assessing symptoms effect in everyday, daily activities interference, so informing us the impact of benign hyperplasia of prostate. This can be used with International prostate symptom score.(American Urology Association 2010).

Symptom quantification is useful for diagnosis, determination of disease severity and monitoring of benign prostatic hyperplasia. International Prostate Symptom Score is the standard got from the American Urological Association Symptom Index published in 1992.[43]

A recent multivariate analysis, found relationship between individual symptoms in theis questionnaire and chronic kidney disease status. They found that obstructive symptoms like poor stream and hesitancy were associated significantly with chronic kidney disease in age.[44] Where as Irritative symptoms had no positive correlation with chronic kidney disease. Moderate to severe Lower urinary tract Symptoms (IPSS > 7) were positively correlated with chronic kidney disease.[9] Kidney failure risks were higher for men with moderate, severe

Lower urinary tract Symptoms compared with men with no or mild Lower urinary tract Symptoms.[11]

Kidney function decreases with age and age significantly correlates with Symptoms of lower urinary tract.[10]

Although symptom score assessment do not directly correlates with chronic kidney disease or can't be used to establish the diagnosis of benign prostatic hyperplasia, it may serve as a basis for symptom severity and management approach to patients with Symptoms of lower urinary tract. Further testing should be considered in patients with an IPSS.

International Prostate Symptom Score (I-PSS)

Patient Name: _____ **Date of birth:** _____ **Date completed** _____

In the past month:	Not at All	Less than 1 in 5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
1. Incomplete Emptying How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
2. Frequency How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
3. Intermittency How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
4. Urgency How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
5. Weak Stream How often have you had a weak urinary stream?	0	1	2	3	4	5	
6. Straining How often have you had to strain to start urination?	0	1	2	3	4	5	
	None	**1 Time**	**2 Times**	**3 Times**	**4 Times**	**5 Times**	
7. Nocturia How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total I-PSS Score							

Score: 1-7: *Mild* 8-19: *Moderate* 20-35: *Severe*

Quality of Life Due to Urinary Symptoms	Delighted	Pleased	Mostly Satisfied	Mixed	Mostly Dissatisfied	Unhappy	Terrible
If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that?	0	1	2	3	4	5	6

Serum creatinine

American Urological Association 2010 Guidelines do not suggest routine serum creatinine measurement in management of benign hyperplasia of prostate. This recommendation is based on the conclusion that renal insufficiency is no more common in men with benign prostatic

hyperplasia than in men of the same age group belonging to general population.

But, the European Association of Urology Guidelines on benign prostatic hyperplasia (2004) and the nephrology-focused NICE (National Institute for Health and Clinical Excellence) guidelines advocated measuring serum creatinine levels in all patients. This is relied on the fact, that bladder outlet obstruction due to benign prostatic enlargement can cause hydronephrosis and renal failure.[23]

Patients with benign prostatic enlargement and renal insufficiency have relatively higher postoperative complications (25% complication rate compared with 17% for patients without renal failure) and mortality goes up to sixfold than those with normal renal function.[37,38, 45] Estimated glomerular filtration rate (eGFR) is a much more reliable measure to define chronic kidney disease and is better than simple serum creatinine measurement.[46]

Urinalysis

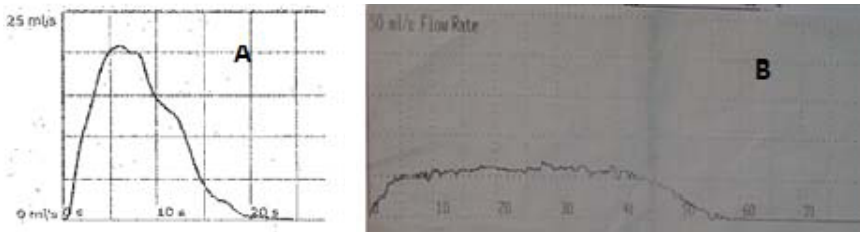
Urinalysis is an inexpensive and simple test that is recommended as a primary evaluation of a patient with suspected benign prostatic enlargement. It is used to rule out urinary tract infection and hematuria. On the other hand, the finding of proteinuria and/or micro- albuminuria can be suggestive of renal failure.

Measuring Total PSA (Prostate specific antigen):

Total PSA is to be offered to patients with more than ten years of life expectancy and in whom measuring Prostate specific antigen will change the management of the lower urinary tract symptoms (AUA 2010 Guidelines). Combining digital rectal examination (DRE) and total PSA measurement becomes the cornerstone of basic screening of prostate. The risks of requiring surgery and developing acute retention is based on measuring PSA and prostatic volume.

Usage of Uroflowmetry (Peak urinary flow rate)

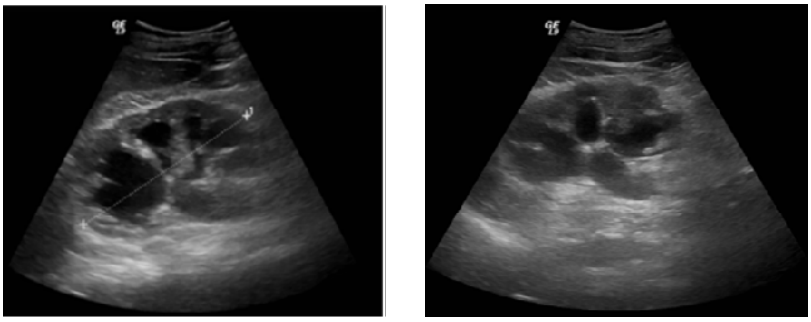
Uroflowmetry is a noninvasive, simple urodynamic test that allows us to have a objective evaluation of the patient micturiating pattern. Although uroflowmetry is an non specific evaluation tool, the micturition graph may suggest some recognizable patterns (e.g. meatal stenosis, urethral stricture, benign prostatic hyperplasia) and it is a tool which is reproduced to quantify the urinary stream strength. It is a useful preoperatively. Peak urinary flow rate (PFR), or Qmax, predicts surgical outcome – by suggesting that, in patients with a preoperative Peak flow rate more than 15 mL/s have poorer outcomes than patients with lesser peak flow rate. It is also an independent predictor for chronic kidney disease rather than reported Lower Urinary Tract Symptoms by standardized questionnaires.[13,9]



Uroflowmetry. A) Normal patient; B) BENIGN PROSTATIC HYPERPLASIA patient

Ultrasound of kidney,ureter, bladder :

Patients with an elevated serum creatinine level, increase post-void residual urine volume are candidates requiring ultrasound of kidney.[47] Renal ultrasonography has many advantages over intravenous urography (IVU) for upper urinary tract. It can measure and evaluate bladder, post-void residual urine volume and prostate, better characterization of renal masses if found, no harm of radiation, no side-effects and lower cost.

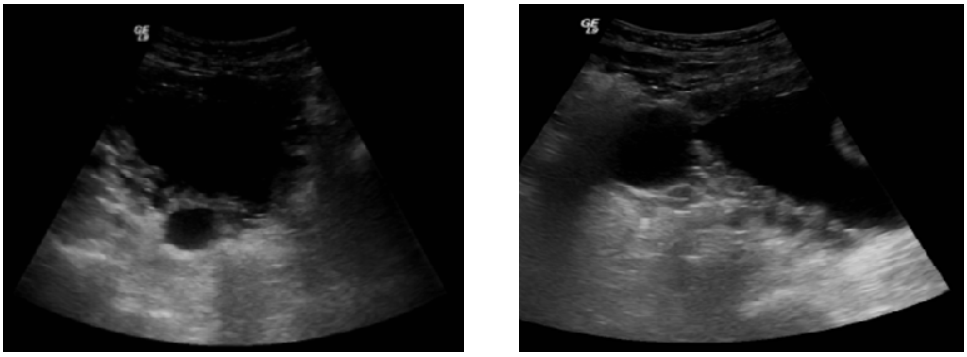


Renal Ultrasound. Two ultrasound scans in benign prostatic hyperplasia patient showing bilateral hydroureteronephrosis (right and left kidney respectively)

Bladder ultrasonography

Measuring bladder wall thickness by transabdominal ultrasound, it is a method to assess bladder outlet obstruction.[49] Measurement of detrusor wall thickness at the anterior wall of bladders having 250 mL

can detect bladder outlet obstruction, if it is more than 2 mm.[50] But it is not recommended by guidelines as it lacks reproducibility.



Bladder Ultrasound. Two ultrasound scans in benign prostatic hyperplasia patient. It is possible to observe the trabecullation, bladder wall thickening and diverticulum

Evaluation of Post-void residual urine:

Post-void residual urine volume can be measured non invasively by transabdominal ultrasonography.[41] It can also be measured by invasive methods like catheterization. Residual urine volumes more than 300 mL affect renal function in cases of benign hyperplasia of prostate.[51,9] Post Void Residual urine more than 100 mL is chronic retention of urine which is associated with chronic kidney disease.[9] Even residual urine volumes less than 100 mL can affect renal function as the presence of residual urine in post void period is related with renal function regardless of the quantity. Thus ultrasonographic evaluation of post-void residual urine is a useful test in the prevention of chronic disease of kidney.

Transrectal ultrasonography of Prostate :

Transrectal ultrasonography of prostate (TRUS) is performed for asssessing prostate size and shape, tissue characterization and carcinoma prostate. There is no relationship between prostatic enlargement measures and chronic kidney disease.[9]

Ultrasonographical Tranabdominal grading of prostatomegaly: Grade I - 3.0 to 3.8 cms, 30 Gms. Grade II - 3.8 to 4.5 cms, 30- 50 Gms. Grade III- 4.5 to 5.5 cms, 50- 80 Gms. Grade IV - 5.5. cms, 80 Gms.[3]

Prostate Ultrasound. Prostate transrectal ultrasonography (sagital view)

Cystometry

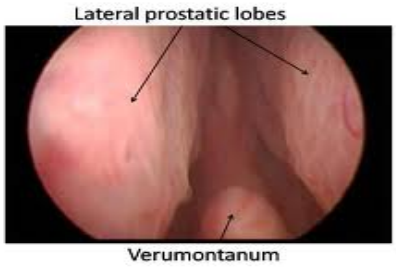
It is usually not a routine examination for benign prostatic enlargement evaluation. However, it can help to identify high bladder pressure, low bladder compliance and detrusor instability that can considerably affect renal function.[9,12]

Use of Urodynamics :

Urodynamics is mainly useful for distinguishing between bladder outlet obstruction and impaired contraction of detrusor. It is performed if it is going to affect the therapeutic decision. Patients with history of neurologic diseases known to affect bladder or sphincter and patients with bothersome symptoms with peak flow rate more than 15 ml/s may benefit.

Urethrocystoscopy

Urethrocystoscopy is not to be done routinely but is optional if invasive treatment is contemplated.[41,43] This also can confirm, other causes of outflow obstruction, sametime eliminating intravesical pathologies.



Cystoscopic view showing enlarged lateral lobes of prostate and verumontanum

Aspect of Treatment :

Patients with mild symptoms are managed by watchful waiting, patients with moderate symptoms should receive pharmacotherapy and patients with severe bother benefit the most from surgical management.

So, a man with preoperative IPSS more than17, has 87% chance of having symptom reduction.[52]

We have to identify a group of patients who are at increased risk of progression (e.g. age, symptoms, Prostate specific antigen level, Qmax, volume of prostate and post-void residual urine). Here we have to give early preventive treatment.[53,50] Because, a higher frequency of kidney failure in patients presenting for prostate surgery than for non prostate surgery has been shown, and several studies have shown improvement in kidney function after prostatectomy.[21]

Emergency situations :

Patients who present to the casualty, with bladder outlet obstruction and high serum creatinine, they should first be put in a urethral catheter and subsequently they need to be evaluated to distinguish between, whether it is an acute and chronic renal failure. Hospitalization is required in these cases. If hydroureteronephrosis and azotaemia persists despite decompressing the bladder, we should suspect an ureterovesical junction obstruction and the next step would be bilateral percutaneous nephrostomy or bilateral double J stents if possible, these are done for temporarily drainage. Patients further may need urgent hemodialysis. Ureteroneocystostomy after a prostate ablation may be adequate for definite ureterovesical junction obstruction resolution.

Benign prostatic hyperplasia - Medical treatment :

Medical approaches are not used to treat if complications are associated with benign prostatic hyperplasia (one of them is chronic kidney disease). They are used for Lower urinary tract symptoms relief and for preventing the progression of benign prostatic hyperplasia (especially 5 alpha reductase inhibitors - 5-ARI).It is also useful in preventing benign prostatic hyperplasia complications such as chronic kidney failure. However, they can't revert chronic kidney disease secondary to benign prostatic hyperplasia.

Surgical treatment

Surgical treatment is mainly offered to men developing complications from benign hyperplasia of prostate. Health Care Policy and Research agency and International Consensus Guidelines, recommend surgery if patient has

1. refractory urinary retention (patient failing atleast one catheter removal attempt)
2. Following conditions secondary to benign prostatic hyperplasia- recurrent Urinary tract infection, bladder stones, recurrent gross hematuria, renal insufficiency, large bladder diverticula.[40]

Some studies suggest that dialysis dependent patients may recover renal function up to a year after prostatic surgery. Here efforts should be made to identify and treat benign prostatic hyperplasia in patients under dialysis.

Erectile dysfunction in about 4% to 10% and urinary incontinence in 0.5% to 1.5% can develop in postoperative period.[54,42] Recurrence of benign hyperplasia of prostate following surgery at five years is about 2% to 10%.

Standard surgical procedures

Transurethral resection of the prostate is the standard and gold standard in the sugery for Benign hyperplasia of prostate. Other procedures are compared against this.[55]

Complications of Transurethral resection of Prostate :[56]

Patients with renal failure undergoing procedure need attention as the complication rates increase in this group including mortality. The complications of TURP are enumerated as follows :

Type of complication	Early		Intermediate		Recent
	Mebust 1989	Doll 1992	Haupt 1997	Borboroglu 1999	Kuntz 2004
Technical complication (%)					
Clot retention	3.3	11.0	1.9	1.3	5.0
Bleeding & transfusion	6.4	22.0	2.2	0.4	2.0
TUR-syndrome	2.0	n.a.	0.3	0.8	0.0
Capsular perforation	0.9	10.0	n.a.	n.a.	4.0
Hydronephrosis	0.3	n.a.	0.0	0.0	0.0
Epididymitis/UTI	3.9	25.0	1.6	4.0	4.0
Urosepsis	0.2	3.0	0.2	0.0	0.0
Failure to void	6.5	3.0	n.a.	7.1	5.0
Incontinence	n.a.	38.0	0.3	n.a.	1.0
Associated morbidity					
Cardiac arrhythmia	1.1	n.a.	0.4	1.3	n.a.
Myocardial infarction	0.05	0.5	0.2	0.2	0.0
Pulmonary embolism	n.a.	n.a.	0.1	n.a.	0.0
Pneumonitis	n.a.	n.a.	0.2	n.a.	0.0
COPD	0.5	n.a.	0.1	n.a.	n.a.
Deep vein thrombosis	n.a.	n.a.	n.a.	0.0	0.0
Mortality	0.23	0.8	0.1	0.0	0.0

n.a. = not available.

Intra-operative and peri-operative complications of TURP[56]

Authors	N	Incontinence (%)	Re-TUR (%)	Impotence (%)	Stricture (%)
Early					
Zwergel 1979	232	11.4	n.a.	n.a.	4.4
Doll 1992	388	9.0	1.5	24[a]	n.a.
Intermediate					
Zwergel 1995	214	3.2	n.a.	n.a.	3.9
Horninger 1996	1211	7.6	n.a.	n.a.	5.6
Hammadeh 1998	52	0.0	4.0	11.0	8.0
Gallucci 1998	80	3.8	0.0	5.0	3.8
Gilling 1999	59	3.2	6.6	8.2	9.8
Borboroglu 1999	520	n.a.	2.5	2.1	3.1
Recent					
Kuntz 2004	100	5.0	3.0	10.5	2.2
Muzzonigro 2004	113	1.8	n.a.	n.a.	3.6

n.a. = not available.
[a] 22% preoperative impotent.

Main late post-operative complications of TURP[56]

Complications

Bleeding

Arterial bleeding is more noticed in cases with preoperative infection, retention of urine due to gland getting congested. Antiandrogen preoperatively can help in decreasing bleeding. Venous bleeding due to perforation of capsule and opening of venous sinusoid during surgery. The amount of intraoperative bleeding usually depends on size of prostate gland and amout resected.

Extravasation

If bladder neck division occurs or capsule perforated, extravasation occurs. It is usually extraperitoneal, but if bladder injured or diffusion occurs in large volume, it can become intraperitoneal.

Injury to Ureteric orifices :

It can occur during large median lobe resection where it becomes difficult to identify ureteric orifice. Treatment depends on severity.

External sphincter injury :

Injury occurs mostly at ventral area (at 12’0 clock postion) because we cannot visualize verumontanum. But if verumontanum is already resected, the sphincter injury risk increases.

Postoperative complications[57]

Bladder tamponade

Evacuation of clots due to recurrent or persistent bleeding or reintervention occurs in 1.3–5% patients. Changing of colour from clear to red intermittently in irrigation suggests arterial bleeding, whereas venous bleeding usually results in a irrigation fluid continuously showing dark red colour.

Infection

The infection rate is usually low .Risk factors for infection :

- Preoperative bacteruria
- Duration of procedure exceeding 70 min
- Tamponade evacuation
- Preoperative stay longer than two days

Retention of Urine :

It occurs in 3–9% of patients. Mostly due to detrusor failure rather than, incomplete resection. It is advised not to go for resurgery till prostatic fossa is healed, exception is if transrectal ultrasound showing significant tissue like ventile effects.

Incontinence postoperatively

Early incontinence occurs in 30–40% of patients, but late iatrogenic stress incontinence occurs in less than 0.5%.

Urethral stricture[58]

Literature suggests 2.2% to 9.8% occurrence and no relationship to time present. The locations and reasons are

-Meatal strictures occurs due to inappropriate size of the instrument and the diameter of meatus.

-Bulbar strictures are due to inadequate isolation by the lubricant, so monopolar current leaks. We need to apply gel in urethra and along resectoscope shaft. Reapply the gel if procedure is long. Avoid high cutting current. Perform internal urethrotomy if meatus narrowed or stricture present.

Bladder neck stenosis

It is around 0.3% to 9.2% in incidence, more with lesser than 30g gland resection. Prophylactic bladder neck incision while concluding procedure may decrease incidence. Once it develops, treatment is by incision by laser or electrical current.

Retrograde ejaculation

Avoiding of tissue around bladder neck leads to reduced incidence. Especially in younger patients, try medical treatment. Transurethral incision leads to reduced incidence.

Erectile dysfunction

Around 3.4% to 32% develop erectile dysfunction. High frequency current applied close to the capsule damages neurovascular bundle.

Recurrent BENIGN PROSTATIC HYPERPLASIA

Usually due to insufficient resection or natural course of disease, but it is lesser with TURP than Transurethral microwave therapy and Trans urethral needle ablation.

TUR syndrome[59]

It is around 2% risk that TUR-syndrome develops. It is due to fluid intoxication, serum sodium level less than 130nmol/L. Large glands, venous sinus opening, prolonged resection time, smoking history increase risk.

Post-obstructive diuresis

Marked natriuresis along with water excretion characterize this disorder. In addition other serious electrolyte disorders such as hypokalemia, hyponatremia and hypomagnesemia can occur.

The etiology is multifactorial, related to fluid and urea accumulation in obstruction and tubular resistance to aldosterone and antidiuretic hormone.

Treatment by fluid replacement with 0.45% saline, at a rate slightly less than urine output and replacement of electrolytes as they are needed.

Renal recovery[61,62]

Complete or prolonged partial urinary tract obstruction leads to tubular atrophy and eventually irreversible renal injury. Prognosis after relief of obstruction is depends on duration, severity of obstruction. Complete recovery of glomerular filtration rate occurs if relieved within one week, but little or no recovery, occurs after 12 weeks.

However, measurement of the Glomerular Filtration Rate probably overestimates the true degree of recovery. In a rat model in which complete unilateral ureteral obstruction was induced for 24 hours, approximately 15 percent of nephrons were nonfunctional as late as 60 days after release, a presumed reflection of irreversible injury .Despite this nephron loss, the Glomerular filtration rate can return to normal because of hypertrophy and hyper filtration in the remaining functional nephrons. It is likely that a similar process of compensatory hypertrophy occurs in human obstruction, as it has been demonstrated in other diseases such as lupus nephropathy. The course of partial obstruction is less predictable. It clearly depends on the severity and duration of the

obstruction, as well as other potential complicating factors, such as hypertension, infection, or preexisting renal disease.

Functional recovery[61,62]

Radionuclide scanning and renal ultrasonography have been used in an attempt to predict the likelihood of functional recovery. Adverse prognostic findings thought to be indicative of severe and usually irreversible disease include total nonvisualization on renal scan and marked cortical thinning on ultrasonography.

But, these findings may not be useful in the individual patient, since their presence does not preclude substantial return or even near normalization of the GFR following release of the obstruction

Most of the functional recovery will usually be seen in the first 7 to 10 days after relief of the obstruction. However, some patients with severe renal failure may, after the obstruction is relieved, require dialysis for a period of weeks until sufficient improvement occurs to allow dialysis to be discontinued.

Only partial recovery is seen in this setting with the plasma creatinine concentration generally stabilizing at a value above 3 mg/dL (264 μmol/L).

MATERIALS AND METHODS

Study Design : Prospective study

Duration : August 2012 to Dec 2013

Setting : Govt. Stanley Medical college and Hospital, Chennai,

This is a clinical study of 40 cases of Benign prostatic hyperplasia in normal and in patients with non dialysis requiring renal failure who underwent surgical therapy-TURP.

PATIENT SELECTION

INCLUSION CRITERIA

All patients with Non dialysis requiring Renal Dysfunction associated with Benign Enlargement of Prostate in the Department of Urology in our institute.

EXCLUSION CRITERIA

- Histologically proven malignant prostatomegaly
- Patients undergoing open prostatectomy
- Patients with end stage renal disease requiring hemodialysis.

DATA COLLECTION

This is a clinical study of 40 cases of BPH who underwent surgical therapy – Transurethral resection of prostate in our institute, out of which 20 patients presented with elevated renal parameters.

The screening was done by selecting all the patients presenting with BPH (350 patients) at our institute during the study period, out of which patients who had associated renal failure on the basis of serum creatinine value were selected. Serum creatinine level of greater than 1.4mg/dl was taken as criteria to determine the presence of renal failure. Among the patients who had BPH with renal failure cases which satisfied the inclusion criteria (20 patients) were selected and rest of the cases i.e. cases which required dialysis, prostatic malignancy, causes of obstructive uropathy other than BPH were filtered out.

All the patients in the study group presented to our institute with severe obstructive voiding symptoms including retention of urine who underwent urethral catheterization and residual urine were measured. All the patients underwent ultrasound abdomen, some of the patients before and some after catheterisation.

Serum Prostate Specific Antigen (PSA) levels were done only in patients with suspicious finding on digital rectal examination.

Urodynamic study was done in selected patients to rule out neurogenic bladder. After the stabilization of renal function (RFT) all the patients underwent diagnostic Cystoscopy followed by TURP. Indwelling 20 Fr three way Foley's catheter was inserted which was removed on 4^{th} post operative day. Patients who went for retention after catheter removal were re catheterised and check cystoscopy done later to rule out the possible obstructive causes of retention. Patients who voided successfully were discharged. Histology of the resected prostate confirmed benign prostatic hyperplasia in all cases.

In the post operative period Serum creatinine estimation was done on 2^{nd},7^{th} day and at 6weeks. Ultrasound was done 6 weeks post operatively.Patients with non dialysis requiring renal insufficiency and normal patients were grouped as groups 1 and 2, respectively.Patient age, comorbid diseases, IPSS, residual urine volume, prostate volume, urea, creatinine (at presentation, post cathetrisation), Sodium, potassium, hemoglobin levels recorded preoperatively. Bleeding time and clotting time done preoperatively in all patients to rule out any coagulation abnormality.If any patient in group 1 shows a drop of s.creatinine below 1.4 in the post cathetrisation setting, they were shifted to group 2 along with normal patients.

Transurethral resection of prostate was performed using a 24 French Storz resectoscope and 1.5% glycine solution or with normal saline (bipolar). Regional anaesthesia was employed. Early postoperative values of hemoglobin, Na, K, and creatinine levels which were measured 24 hours after the operation were recorded. The need for blood transfusion and presence of a TUR syndrome were also evaluated. The catheters of the patients were removed in 4^{th} Postoperative day after the urine became clear.

DATA ANALYSIS:

STATISTICAL METHODS APPLIED

Frequencies

The Frequencies procedure provides statistics and graphical displays that are useful for describing many types of variables. The Frequencies procedure is a good place to start looking at your data.

Descriptives

The Descriptives procedure displays univariate summary statistics for several variables in a single table and calculates standardized values (z scores). Variables can be ordered by the size of their means (in ascending

or descending order), alphabetically, or by the order in which you select the variables (the default).

Chi-Square Test

The Chi-Square Test procedure tabulates a variable into categories and computes a chi-square statistic. This goodness-of-fit test compares the observed and expected frequencies in each category to test either that all categories contain the same proportion of values or that each category contains a user-specified proportion of values.

Crosstabs (Contingency coefficient test)

The Crosstabs procedure forms two-way and multiway tables and provides a variety of tests and measures of association for two-way tables. The structure of the table and whether categories are ordered determine what test or measure to use.

Paired samples t test

The Paired-Samples T Test procedure compares the means of two variables for a single group. It computes the differences between values of the two variables for each case and tests whether the average differs from 0.

Independent-Samples T Test

The Independent-Samples T Test procedure compares means for two groups of cases. Ideally, for this test, the subjects should be randomly assigned to two groups, so that any difference in response is due to the treatment (or lack of treatment) and not to other factors.

All the statistical calculations were done through SPSS 16.0 (2007) for windows.

Multinomial Logistic Regression

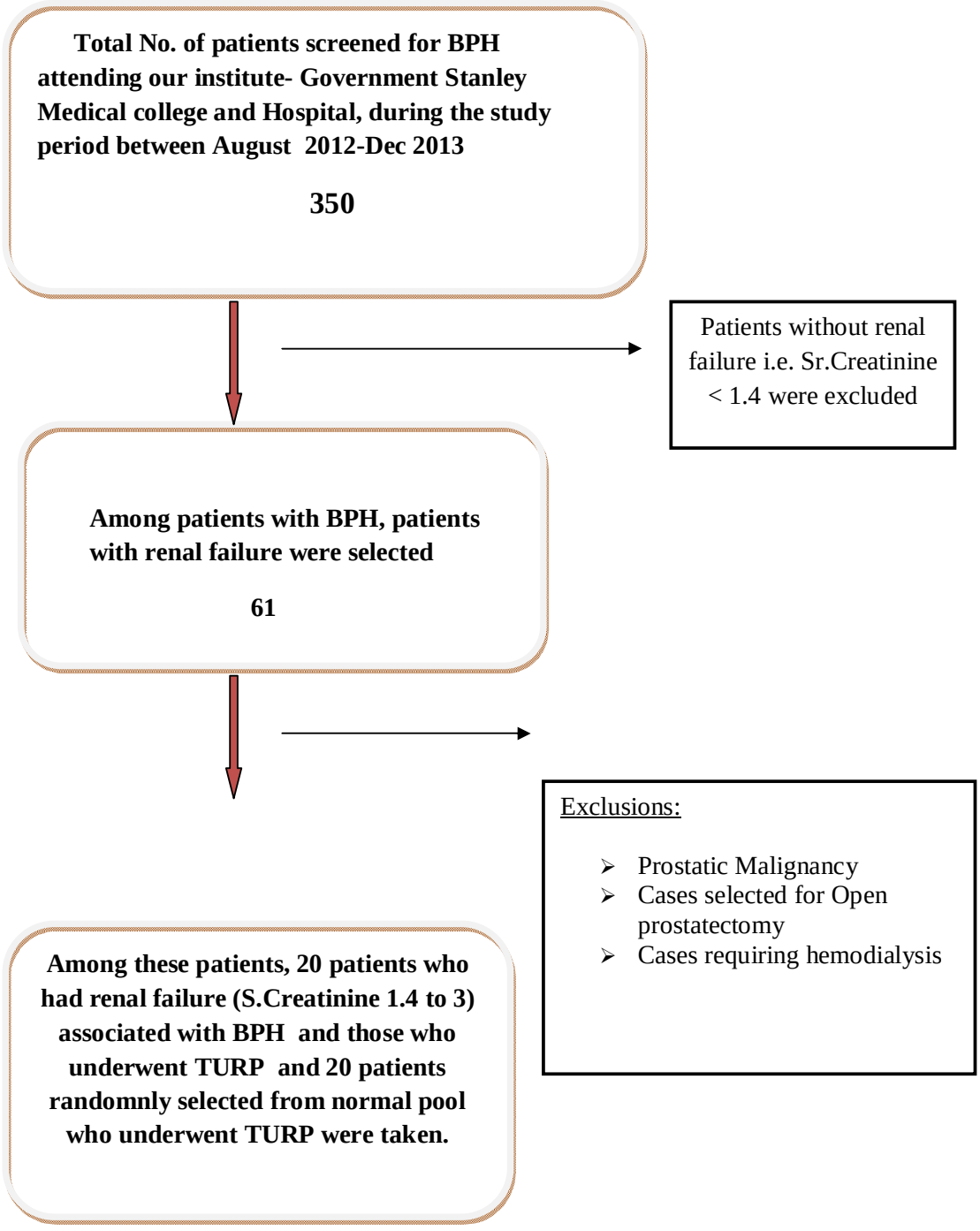
Multinomial Logistic Regression is useful for situations in which you want to be able to classify subjects based on values of a set of predictor variables.

Pearson's correlations coefficient

The Bivariate Correlations procedures computes Pearson's correlations coefficient. Correlations measure how variables or the Rank orders are related.

The statistical operations were done through SPSS 16.0 (2007) for windows and EPIINFO software

OBSERVATION AND RESULTS:



In our study total of 350 patients at our institute were screened during the study period and 20 of those patients who satisfied the inclusion criteria were included in the study group. The observation and results of the study were as follows.

1.Descriptive Study

Table 1.0. Age Distribution of study population : Group 1

Age (in yrs)	**No. of patients**	**Percentage**
51-60	5	25%
61-70	11	55%
71-80	4	20%
81-90	0	0%
Total	20	100%

	Group	N	Mean	Std. Deviation	P value
Age in years	Group I	20	65.00	5.620	0.620
	Group II	20	64.00	6.959	

Age distribution pattern in Group 1 patients

In a total of 20 patients in Group 1, the youngest was 55 years and oldest was 75 years with a mean age of 64 years and standard deviation of 5.62 with the predominant age group 61-70 years.

In Group 2,

Age (in yrs)	**No. of patients**	**Percentage**
51-60	8	40%
61-70	7	35%
71-80	5	25%
81-90	0	0%
Total	20	100%

Age distribution pattern in Group 2 patients\

In Group 2, the youngest patient was 52 years and oldest patient was 76 years with a mean of 64 years with a standard deviation of 6.95.

Group 1

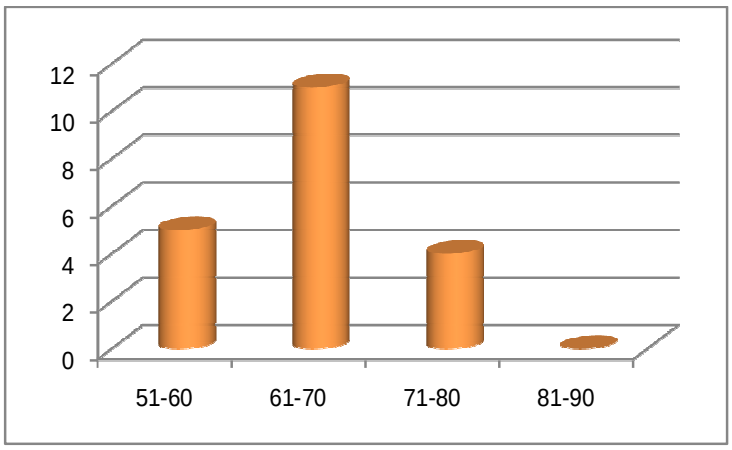


Figure 1. Frequency distribution of population in Group 1

In Group 2

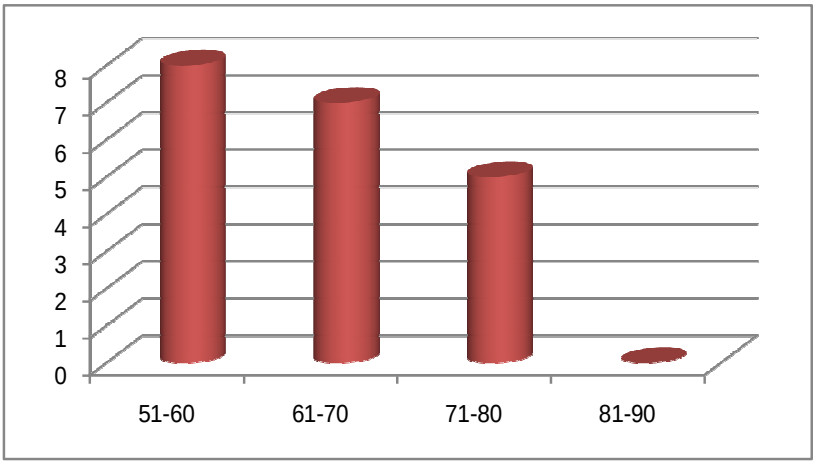


Figure 1. Frequency distribution of population in Group 2

Symptom score and Quality of Life					
	N	**Minimum**	**Maximum**	**Mean**	**Std. Deviation**
IPSS Group 1	20	20	24.95	1.986	.444
Group 2	20	20	24.65	1.899	.425

Table 1.3. Symptom score

All the patients had severe IPSS (majority had obstructive symptoms) with a mean score of 24.95 in Group 1 and 24.65 in Group 2,minimum and maximum score of 20 and 25 respectively with a standard deviation of 1.98. On analysis of IPSS score it was found that mean obstructive symptoms score was 14.3 as compared to the mean irritative symptoms score of 10.5.

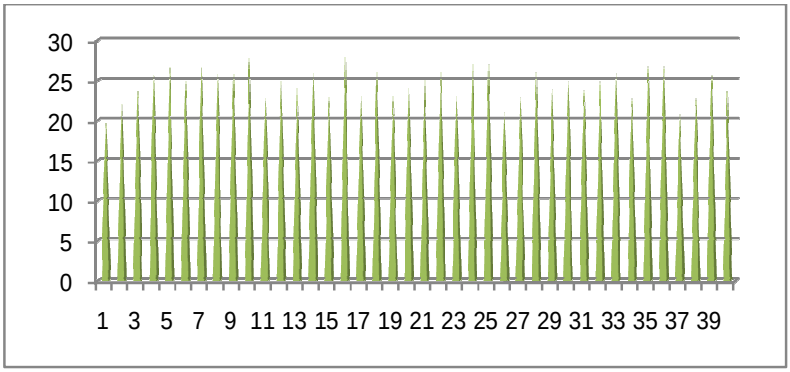


Figure 3. IPSS score in this study population

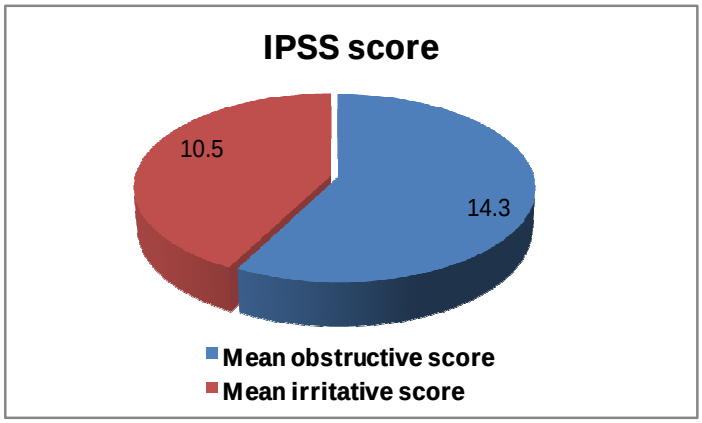
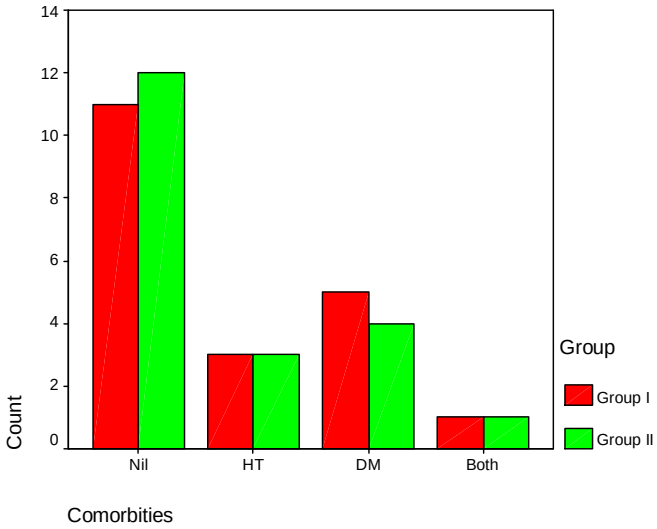


Figure 4. IPSS score (obstructive vs. irritative score)

Comorbities associated with groups

In Group 1, out of 20 patient who presented, nine people had comorbidities, where as in goup two eight people had comorbidities. The difference between these two groups was not significant.



Correlation between comorbidities like hypertension, diabetes in both groups

			Group		Total
			Group I	Group II	
Comorbities	Nil	Count	11	12	23
		% within Comorbities	47.8%	52.2%	100.0%
		% within Group	55.0%	60.0%	57.5%
	HT	Count	3	3	6
		% within Comorbities	50.0%	50.0%	100.0%
		% within Group	15.0%	15.0%	15.0%
	DM	Count	5	4	9
		% within Comorbities	55.6%	44.4%	100.0%
		% within Group	25.0%	20.0%	22.5%
	Both	Count	1	1	2
		% within Comorbities	50.0%	50.0%	100.0%
		% within Group	5.0%	5.0%	5.0%
Total		Count	20	20	40
		% within Comorbities	50.0%	50.0%	100.0%
		% within Group	100.0%	100.0%	100.0%

Correlation of comorbidities both within the group and between the two groups

Ultrasound prostate size :

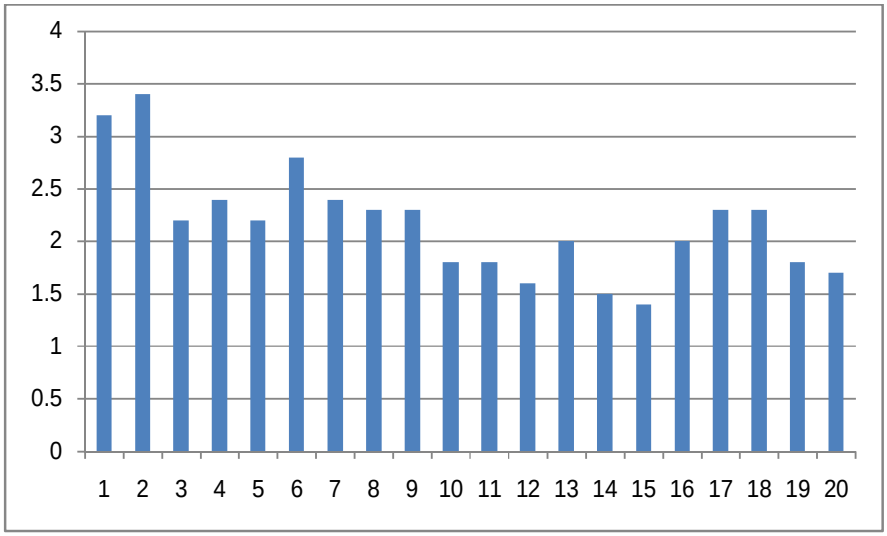
Comparing the prostate size by ultrasound in both groups, the mean prostate volume was 46cc in group 1 with a standard deviation of 12.47 and 42 cc in group 2 with a standard deviation of 14.19.

Residual urine measurement :

When we compare the residual urine in both groups, residual urine in patients in group1 was in the range of 165 ml and in group 2 it was in the range of 136ml.

S.creatinine at presentation :

In group 1, patients who presented with elevated s.creatinine values, the following values were recorded at presentation



Picture representing levels of S.creatinine in patients during presentation

It ranged from 3.4 to 1.7, with a mean value of 2.170 and a standard deviation of 0.5. All these patients underwent catherization accordingly, then their s.creatinine was recorded once it got stabilized. Whereas in group 2, the mean serum creatinine was around 0.925 with a standard deviation of 0.1.

Preoperative Serum creatinine :

Once the serum creatinine values stabilized, the readings were recorded. Out of 20 patients who presented with elevated renal parameters, (patient with s.creatinine more than 1.4) five patients subsequently showed fall in serum creatinine below 1.4, hence were considered along with normal patients in group 1. Because of this, the subsequent serum creatinine values of rest of 15 patients stabilized at a mean of 1.7 with a standard deviation of 0.3. Interestingly patients whose serum creatinine which stabilized at a value of more than 1.4, some of them had coexistent diabetes, hypertension. This might be an explanation that these patients have developed preexisting renal disease which was worsened by their developing benign prostatic hyperplasia.

	Group	N	Mean	Std. Deviation	Std. Error Mean
Preop S.creatinine	Group I	15	1.747	.3021	.0780
	Group II	25	.960	.1258	.0252

Table showing mean creatinine levels in preoperative period

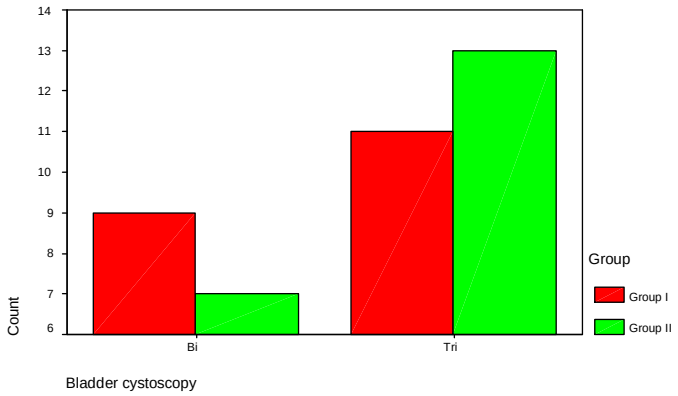
Comparison of preoperative sodium, potassium values in both groups:

Preop Na values	Group I	15	138.13	1.642	.424
	Group II	25	138.48	1.711	.342
Preop K levels	Group I	15	4.693	.3327	.0859
	Group II	25	4.604	.1098	.0220

Table showing mean preoperative sodium and potassium levels in both groups

As depicted above, in group 1, the mean sodium value was 138, whereas in group 2, the mean sodium value was 138.48. There was no significant difference in these two groups. Similarly, the mean preoperative potassium levels in both groups were not significantly different.

Cystourethroscopy findings :



Cystourethroscopy findings of prostate and bladder in both groups.

On cystourethroscopy, out of 40 patients 16 had bilobar and 24 had trilobar enlargement.

CYSTOSCOPY – BLADDER TRABECULATIONS:

Group 1: In group 1, out of 15 , 10 patietns had grade 2 trabeculations, rest with grade 3 trabeculations**.**

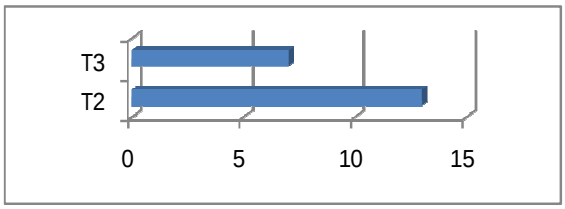


Figure 10. Cystoscopy-Bladder Trabeculations in Group 1

Group 2 : In group 2, out of 20, 17 patients had grade 2 trabeculations, rest with grade 3 trabeculations.

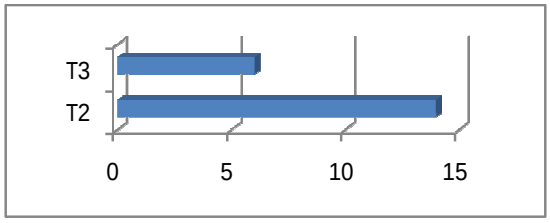


Figure 10. Cystoscopy-Bladder Trabeculations in Group 2

Resection time during surgery :

The mean resection time in group 1 was 53 minutes , with a standard deviation of 8.8 minutes. In group 2, the resection time was 56 minutes mean value with a standard deviation of 9.7 minutes.

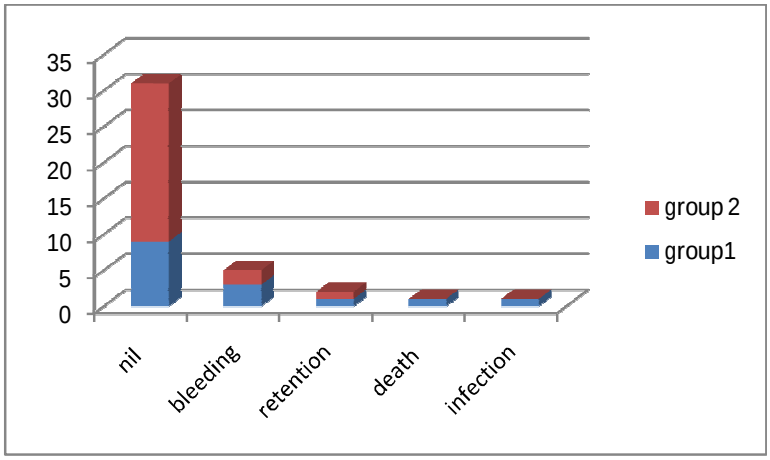


Figure 11. Frequency distribution of complications

As mentioned above in group 1, out of 15 patients, three people had bleeding, one had retention, one perioperative death due to myocardial infarction was recorded, one patient had infection.In all the patients with bleeding as complication, blood transfusion was given. Whereas in group 2, two people had bleeding out of 25 patients studied. (8% compared to 20% in group 1). When we taken retention, three people have developed in group 2. In case of infection, none had developed, compared to one patient in renal failure group. Death was not noted in group 2, whereas one recorded in group1.

Post operative values :

The mean postoperative sodium value in group 1 was 137.53 with a standard deviation of 1.995, where as in group 2 it was 137.92. So, not much of difference were noted. Similarly, post operative potassium

values were in the range of 4.8 in group 1 with a standard deviation of 0.3 compared to group 2 with a mean of 4.7.

Serum creatinine levels in group 1 post surgery :

Mean serum creatinine level in group 1, which was measured at 2nd post operative day was 1.6 with a standard deviation of 0.2. Same group who had serum creatinine measured at 14th Post operative day had a mean of 1.56, and at 6 weeks maintained at a mean of 1.56. This shows there was a drop in serum creatinine values post surgery.

Analysis of various factors :

Analysis of variation in serum sodium levels and potassium in preoperative and postoperative setting :

GROUP 1 :

	Mean	N	Std. Deviation	Std. Error Mean
Preop Na values	138.	15	1.642	.424
Post op Na values	137.53	15	1.995	.515
Preop K levels	4.693	15	.3327	.0859
Post op K levels	4.800	15	.3359	.0867

Table showing preoperative and postoperative sodium and potassium levels in Group 1 patients

As depicted in the above table the preoperative sodium and postoperative sodium in group 1, was 138 and 137.53, respectively. Similary with potassium it was 4.6 and 4.8 respectively.

When analysed with paired samples test, both of them were insignificant, meaning no significant difference in the values noted in the elevated renal parameters group.

GROUP 2:

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Preop Na values	138.48	25	1.711	.342
	Post op Na values	137.92	25	1.730	.346
Pair 2	Preop K levels	4.604	25	.1098	.0220
	Post op K levels	4.708	25	.1498	.0300

Table showing preoperative and postoperative sodium and potassium levels in Group 2 patients

Similarly, in group 2 the difference in the values in the preoperative and postoperative period was not significant.

Serum creatinine values preoperative period and after 6 weeks post transurethral resection comparison :

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Preop S.creatinine	1.714	14	.2852	.0762
	S.creatinine 6 weeks	1.5643	14	.25603	.06843

Table showing mean S.creatinine improvement in patients in preoperative period and post operative period

	Paired Differences					t	df	Sig.
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				P value
				Lower	Upper			
Preop S.creatinine - S.creatinine 6 weeks	.1500	.10919	.02918	.0870	.2130	5.140	13	.000

As mentioned above , the fall in serum creatinine between the time of presentation and post operative period, was significant. This implies that Transurethral resection has made a positive outcome in this group of patients.

DISCUSSION

Benign Prostate Hyperplasia (BPH) is a common disease in ageing men with a prevalence of 50% above 50 years and increasing up to 80% in men above 80 years. In our institute it was observed that many patients who presented with obstructive Lower Urinary Tract Symptoms secondary to BPH had associated renal failure.

While the underlying mechanism for developing renal failure associated with BPH is multifactorial and co-morbid factors such as diabetes, hypertension, etc in elderly men may contribute to renal impairment, we wanted to evaluate the morbidity and mortality and outcome in patients undergoing transurethral resection with nondialysis requiring renal failure.

Despite the many possible causes of renal failure in elderly patients, the common causes were BPH (38%), neurogenic bladder(19%), obstructive pyelonephritis (15%). If we have to go by the natural history of the disease progression of BPH 13.6% of patients who presented to undergo TURP were in renal failure.

Attending to high prevalence of BPH in older men with renal failure it is invaluable to take into consideration the relationship between these two clinical entities. However, despite the high prevalence of renal

failure and BPH in elderly men, there is very limited knowledge on the association between these two conditions and it is important to discuss the relationship between BPH and renal failure.

We decided to study the association of BPH with renal failure and role of TURP which is the gold standard surgery for BPH and its associated outcome, morbidity and mortality. A similar study determining the role of TURP in management of BPH Presenting with Renal Failure was taken up by Thomas et.al.[55]

In our study the mean incidence of BPH with renal failure was 12.5%, (61/350patients) which was comparable with the other studies. The AHCPR BPH Guidelines report a mean of 13.6% of renal failure. In another study there was a reported incidence of 11% in patients with renal failure secondary to BPH.[55]

Since renal failure is a multifactorial process making it harder to investigate the true influence of BPH on renal failure, the study was designed such that patients who had renal failure associated with benign hyperplasia of prostate, after excluding cases of prostatic malignancy, other causes of obstructive uropathy,as found that only 20 out of these 350 patients satisfied our inclusion criteria.

In our study the average age of presentation was 65 yrs and the majority of patients in their sixth decade. Both diseases are extremely

common among aging male, leading some to suggest that it is a natural concomitant of aging.[8]

It is known that age is a independent risk factor for renal insufficiency irrespective of medical co-morbidities due to age related nephron loss and age is also risk factor for the development of BPH. Both diseases are extremely common among aging male, leading some to suggest that it is a natural concomitant of aging.[8]There was a cross-sectional association between signs and symptoms of bladder outlet obstruction and chronic kidney disease in community-dwelling men.[(9)] In contrast, a population-based study from Austria there was no significant association between degree of LUTS and GFR after adjusting for age in this cross sectional study.[(10)] In our study severity of LUTS did affect the final outcome, however this could be confounded by other risk factors such as age, severity of renal failure, degree of bladder dysfunction etc.

More recently a cross-sectional survey in Spain of 2,000 randomly sampled men who were 50 years or older showed a 2.4% prevalence of self-reported renal failure related to a prostate condition (9% reported renal failure from any cause).[63,9] Another study[21] showed that men presenting for prostate surgery had a 7.7% prevalence of renal failure compared to a 3.7% prevalence in age matched men presenting for nonprostate surgery.

Among patients who presented to us 15 patients (50%) were in acute retention of urine, 30% were in chronic retention and the remaining had severe obstructive voiding symptoms.

In acute retention of urine there is rapid progression of renal failure due to defect in renal tubular function and most of the cases recover well following catheterisation unless they are in acute on chronic retention of urine where in patients take a longer time for their renal functions to recover while few cases may go into a state of refractory renal failure.

All the patients had an IPSS suggestive of severe lower urinary tract symptoms with bothersome quality of life score being unhappy and it was also observed that major contribution in their IPSS were from obstructive symptoms as compared to irritative voiding symptoms.

In BPH, symptoms results from the direct bladder outlet obstruction (BOO) from enlarged tissue (static component) and the increased smooth muscle tone and resistance within the enlarged gland (dynamic component). These physiologic issues reflect in voiding dysfunctions, that significantly affects the health and quality of life of many older men.

Although signs and symptoms of BPH are normally present, there are a significant number of patients that are relatively asymptomatic[35]

(without significant voiding dysfunction), but can present primarily as clinical sequel of renal insufficiency –uremia; with nausea, vomiting and mental status changes – and analytical changes –electrolyte disturbances (hypercalcemia and nonanion gap acidosis).

Older patients with voiding dysfunctions caused by chronic urinary obstruction might present with hypertension due to hypervolemia in the case of bilateral obstruction or increased renin release. Hypertension, on other hand can be itself the sole cause of renal failure.[35]

In our study serum creatinine (SC) and creatinine clearance(Cr.Cl) were taken as criteria for defining renal insufficiency. Serum creatinine of 1.4mg/dl were taken as the cut-off, a value above which were included in the study. The routine measurement of serum creatinine levels is not indicated in the initial evaluation according to the AUA Guideline Management of BPH (AUA 2010 Guidelines). This recommendation is based on the conclusion that baseline renal insufficiency appears to be no more common in men with BPH than in men of the same age group in the general population.In our study routine screening of serum creatinine not only identified patients with renal failure but also was found to be a significant risk factor for causing complication.

Coming to the comorbidities, nine patients out of 20 people enrolled under elevated renal parameters group had hypertension,

diabetes or both. This was not significant when compared to the normal group.

We grouped patients with elevated renal parameters and 20 patients were enrolled into group 1. Selected patients underwent cathetrisation, post catheterisation serum creatinine values settled. All our patients who came to our outpatient department underwent urinary diversion in the form of urethral catheterization, subsequently underwent TURP after stabilisation of renal functions & once the patient was medically fit to undergo TURP. In five patients serum creatinine values normalized below 1.4, hence they were shifted to group 2. Many of the patients presented with marginal elevation of serum creatinine indicating early renal insufficiency, this could be attributed to severity of symptoms and acute retention of urine forcing them to seek medical attention..

Following TURP the outcome and complications were analyzed. A positive outcome was defined as restoration of normal voiding pattern (as reflected by post void residual urine measurements); improvement in the renal function, and those who did not satisfy above definition were regarded as complications or negative outcome.

It was found that there was a statistically significant (p value=0.000) improvement in serum creatinine and post void residual

urine in the post TURP period suggesting that TURP had a very significant role in modifying these factors.

We further analysed these factors, some patients whose serum creatinine did not touch the baseline, had comorbidites, which might suggest that the patients can have a preexisting renal disease, which was further worsened by benign hyperplasia of prostate.

The mean prostate volume in both groups was not significantly different, both ranging from 42 to 46 cc volume. The resection time was also not very different between both groups ranging from 52-55 minutes. Mean difference in the levels of sodium, potassium were not significant in both groups in preoperative and postoperative period. No incidence of TURP syndrome was noted in our study. In our study there was no association found between volume of prostate gland with final outcome. Previous studies which examined the association between prostate size and renal function gave conflicting results[9] some showing a strict relation between prostate size and GFR[65] but other studies did not.[66] Recent studies were made to relate prostate size and LUTS in BPH. Hassanzadeh et. al,[20] found a significant correlation between urgency and prostate size, which can be considered as predictive factor for the disease and probably a strong link between BPE and CKD.

In other studies, it was shown that patients with BPH and renal insufficiency have much higher postoperative complications (25% complication rate compared with 17% for patients without the condition) and mortality (up to sixfold) than those with normal renal function.[37,38,39]Comparing the complications between two groups, it was found that eventhough complications like bleeding was higher than the normal group including need for blood transfusion, they were not statistically significant. This may be attributable to the small sample size on the study point of view. The use of continuous-flow resectoscopes, nonhemolytic irrigation fluids and decrease in operative time, better anesthetic care due to improving and refining of TUR-P technique in time with the increased number of operations performed probably has had significant positive effects on TUR-P outcomes. This leads to a decrease in complication rate compared to older studies. Death was seen in the elevated renal parameter group, none was recorded in group 2. Death in this patient, had been due to myocardial infarction. Renal failure patients are prone for cardiovascular complications.

In our study there was no association found between amount of residual urine with final outcome. For years it has been well described that large volumes (»300 mL) affect renal function in advanced BPH.[24,9,12] Recent studies, however, demonstrate that the volume of

residual urine (post void) necessary to impair renal function is not that elevated.

LIMITATIONS OF THIS STUDY :

1. Sample size in the elevated parameters group who underwent transurethral resection of prostate is less, which was further reduced by patients attaining normalization of creatinine values post catheterization who subsequently got shifted to group 2.
2. This study was conducted in a group of patients with lesser grades of chronic kidney disease, thereby excluding patients with higher grades of Chronic kidney disease and patients on dialysis. To exactly study the impact of transurethral resection and its complications in these important subgroups of patients also becomes important.
3. All the patients who underwent transurethral resection, procedure was not done by a single surgeon. As it is a teaching institute, variations in technique and time varied between individual surgeons.
4. The fourth limitation was follow up. It was hard to get follow up of patients, who often faultered on timely visits to hospital despite reminders.

CONCLUSION

- The incidence of renal failure associated with benign prostatic hyperplasia in our study was about 12.5%. After excluding patients with prostatic malignancy, patients needing dialysis, the study group constituted 5%.
- The age of presentation was around 64-65 years, which also coincided with increased prevalence of chronic kidney disease in elderly population, who also present with benign hyperplasia of prostate.
- There was no significant variation in preoperative and postoperative levels of serum sodium, potassium levels in patients with nondialysis requiring renal failure (S.creatinine less than 3), when compared to normal patients.
- Complications of transurethral resection in patients who had nondialysis requiring renal failure, was on the higher side. Bleeding as a complication requiring blood transfusion was noted in 20% of patients compared to normal patients. But overall complication rate was not statistically significant. This can be attributed either small sample size. Improvement in instruments like bipolar resection, use of non hemolytic irrigation solutions,

continuous flow resectoscopes, better anesthetic care can be a factor.

- The outcome following TURP was successful with restoration in normal renal function and normal voiding pattern in majority of patients and it was further noted that the successful outcome of TURP in these patients were influenced by various factors such as age, duration of symptoms, severity of lower urinary tract symptoms, severity of renal failure at the time of presentation, The size of Prostate gland had no correlation with the final outcome.

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INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Prospective study of effects of Transcrethral resection of Prostate on morbidity and mortality in patients with non Dialysis requiring renal insufficiency

Principal Investigator : Dr.Amar Needhi Ganesan

Designation : PG in M.Ch(Urology)

Department : Department of Urology
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 31.07.2012 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

03/10/12

MEMBER SECRETARY,
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PROSPECTIVE STUDY OF EFFECTS OF TURP ON OUTCOME,MORBIDITY AND MORTALITY IN PATIENTS WITH NON DIALYSIS REQUIRING RENAL INSUFFICIENCY

Dissertation Submitted to

THE TAMILNADU
DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI

In Partial fulfilment of the regulations for the award of the degree of

M.CH DEGREE EXAMINATION
BRANCH IV - UROLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE
***AND* HOSPITAL**

CHENNAI – 600001

AUGUST – 2014

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ANNEXURE

PROFORMA

1. Name:
2. Age:
3. Sex:
4. Address:
5. Date of admission:
6. Date of discharge:
7. IP NO:

CHIEF COMPLAINTS:

- LUTS
- IPSS
- Acute /chronic retention of urine
- Abdominal pain
- Fever and chills (if there is also a urinary tract infection)
- Uremic symptoms

PAST HISTORY: Any associated systemic diseases

TREATMENT HISTORY/ PREVIOUS OPERATIVE HISTORY

GENERAL PHYSICAL EXAMINATION:

Vitals
Pallor
Edema

SYSTEMIC EXAMINATION:

Per ABDOMEN

Per RECTAL:

EXTERNAL GENITALIA:

NEUROLOGICAL EXAMINATION

OTHER SYSTEMS:

INVESTIGATIONS:

Hb,TC, DC
Urine routine

BT/CT/PT
Blood group
HIV
HbsAg
ECG
Chest X ray

RBS
Blood Urea
Sr. creatinine,Sr.Electrolytes

Imaging KUB:

- Upper Tract
- Bladder
- Prostate
- Pre/Post void
- Other findings

Sr.PSA and Prostate Biopsy(Selected cases)
Urodynamics (Selected cases):

Post urinary diversion	Blood urea	Serum creatinine	Serum electrolytes	USG-Upper tract status
Day				
Day				

OPERATIVE PROCEDURE:

Type of Operative procedure done
Date and time:

Findings

Histopathology

FOLLOW-UP	**1 week**	**2 weeeks**	**6 weeks**
SYMPTOMS			
RENAL FUNCTIONS (S.creatinine)			
POST-OP IMAGING (USG KUB)			

நோயாளிகளுக்கான ஆலோசனை

சிறுநீர் வரும் தாரையில் பராஸ்டேட்டு என்னும் சுரபி உள்ளது. அது பொதுவாக வயதானவர்களுக்கு பெரியதாய் வளர வாய்ப்பு உண்டு.

இதனால் சிறுநீர் கழிப்பதில் சிரமம் மற்றும் அடைப்பு ஏற்பட வாய்ப்பு உண்டு. இதை மருந்தினால் குணப்படுத்தாமல் போகும் பட்சத்திலோ அல்லது அடிக்கடி அடைப்பு, கல், சிறுநீரக பாதிப்பு, சிறுநீர் கிருமி தொற்று, போன்றவை ஏற்படும் போது அதை அகற்ற வேண்டிய கட்டாயம் ஏற்படுகிறது. இத்துடன் வரும் நோயாளிகளில் சிலருக்கு சிறுநீரக பாதிப்பு ஏற்பட்டு உப்பு சத்து உடம்பில் அதிகமாக ஆவது உண்டு. எனவே உப்பு சத்து அதிகமாக உள்ளவர்களுக்கும் உப்பு சத்து சரியான அளவில் உள்ளவர்களுக்கும், பராஸ்டேட்டு சுரபி அகற்றும் போது ஏற்படும் விளைவுகள் பற்றி நான் ஒரு ஆய்வு மேற்கொண்டு உள்ளேன்.

இந்த கண்காணிக்கப்பட்ட மருத்துவ ஆய்விற்கு தாங்களும் பதிவு செய்து தங்களது முழு ஒத்துழைப்பை நல்குமாறு தங்களை அன்புடன் கேட்டுகொள்கிறேன் .

நோயாளிகள் ஒப்புதல்

இந்த நோய், அதற்க்கான பரிசோதனை மற்றும் நடத்தப்படும் ஆய்வை பற்றி முழுமையாக மருத்துவர் விளக்கினார். நான் இந்த ஆய்வில் பங்கெடுக்க முழு மனதுடன் சம்மதம் தெரிவிகின்றேன் .

நோயாளியின் கையொப்பம்

அனுப்புனர்

பெயர் :

தந்தை பெயர் :

முகவரி :

வயது :

பெறுநர்

ஐயா,

நான் மேற்சொன்ன முகவரியில் வசித்து வருகிறேன்.தற்போது ..... தொழில் செய்து வருகிறேன். எனக்கு சிறுநீர் கழிப்பதில் சிரமம், அடைப்பு, அடிக்கடி கழித்தல் போன்ற தொந்தரவிற்காக ஸ்டான்லி மருத்துவமனை வந்துள்ளேன். என்னை பரிசோதித்த மருத்துவர் எனக்கு பராஸ்டேட்டு சுரபி பெரியதாக உள்ளதாகவும், அதற்கு ஆபரேஷன் செய்ய வேண்டிய அவசியத்தை விளக்கிகூறினார். இந்த நோயை பற்றிய சந்தேகங்களை நான் கேட்க விளக்கினார்.இந்த ஆபரேஷன் தன்மை,பக்க மற்றும் பின் விளைவுகளையும் மருத்துவர் விளக்கினார்.

. இந்த ஆய்வினால் எனக்கும், பொதுவாக பராஸ்டேட்டு நோயாளிகளுக்கும் கிடைக்க கூடிய நன்மைகள் எனக்கு எடுத்துரைக்கப்பட்டன. இந்த ஆய்வு குறித்து, நான் எழுப்பிய வினாக்கள் மற்றும் சந்தேகங்களுக்கு மருத்துவர் விளக்கமாக பதிலளித்தார். இவற்றை தெரிந்து கொண்ட நான் எனது சுய நினைவுடன் இந்த ஆய்வில் பங்கேற்க எனது விருப்பத்தின்பேரில் யாருடைய நிர்பந்தமும் இல்லாமல் என் சுய நினைவுடன் இந்த ஆய்வில் பங்கேற்க எனது விருப்பத்தை தெரிவித்துக்கொள்கிறேன். இந்த ஆய்வு, என்னுடைய, மற்றும் என் போன்ற நோயாளியர் நலன் கருதியே செய்யப்படுகிறது என்பதை அறிந்ததால் இதற்கு என்னை ஆட்படதுகின்றேன்.

இந்த ஆய்வு குறித்து முழு விவரங்களை நான் கேட்டு பெற்றுள்ளதாலும், என்னுடைய விருப்பத்தின்பேரில் பங்கு கொள்வதாலும், இது குறித்து எந்த குற்ற முறையீட்டையும் மருத்துவர் மீதோ, ஏனைய மருத்துவ ஊழியர்கள் மீதோ, மருத்துவமனை மீதோ எந்த நிலையிலும் வைக்க மாட்டேன். இதையே என்னுடைய ஒப்புதல் மற்றும் வேண்டுகோள் கடிதமாக ஏற்றுக்கொள்ளுமாறு கேட்டுக்கொள்கிறேன்.

நோயாளியின் கையொப்பம்

பராஸ்டேட்டு ஆபரேஷன் செய்வதால் - உப்பு சத்து சரியான அளவில் மற்றும் அதிகமாக உள்ளவர்களில் ஏற்படும் மாறுபாடு மற்றும் விளைவுகள் பற்றிய ஆய்வு

நோயாளியின் ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை, சென்னை 600001

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் கையொப்பம் :

பங்கு பெறுபவர் இதனை ( ) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது . ☐

நான் இந்த ஆய்வில் தன்னிச்சையாகத்தான் பங்குபெருகிரேன் .எந்த காரணத்தினாலோ எந்த சட்டசிக்கல்களுக்கும் உட்படாமல் நான் இந்த ஆய்வில் இருந்து விலகிக்கொள்ளலாம் என்று அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற் கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்துகொள்கிறேன்.நான் ஆய்வில் இருந்து விலகிக்கிகொண்டாலும் இது பொருந்தும் என அறிந்தேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் , பரிசோதனை முடிவுகளையும், மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட அறிவுரைகளின் படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் ☐

மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதி அளிகின்றேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத, வழக்திர்க்குமாறன நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெருவிப்பேன் என உறுதி அளிக்கிறேன்.

இந்த ஆய்வில் எனக்கு ரத்தம், சிறுநீர், எக்ஸ்ரே, ஸ்கேன், உட்பட அனைத்து பரிசோதனைகளையும் செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்.....இடம்.....தேதி .....

கட்டைவிரல் ரேகை.....

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம்..... ..இடம்..... தேதி.....

ஆய்வாளரின் பெயர் .....

Name	ID	Age(yrs)	IPSS scoring	comorbities	usg prostate size	residual urine	S.creatinine at presentation	Preop S.creatinine	Preop Na values	Preop K levels	Bladder cystoscopy	cys-trab	Resection time in min	Preop Hb levels	Post op hb	Post op Na values	Post op K levels	Post op Creatinine levels	need for blood transfusion	Complications	S.creatinine 2nd day
anbazhagan	40232	55	20	DM	65	180	3.2	2.1	136	4.3	Tri	T2	70	10.8	10	135	4.4	2	Y	bleeding	1.8
Krishna	41292	60	24	HT	60	200	3.4	2.2	138	5.1	Tri	T2	60	10.2	9.2	136	5.3	2	Y	bleeding	2
sambasivan	47195	63	24	DM	61	150	2.2	2	140	5	Bi	T2	40	10.6	9.6	135	5.2	1.9			2
yesudas	39512	62	26		35	180	2.4	2.1	141	5.3	Tri	T2	60	10.8	9.9	136	5.3	1.8		-	2
anthonysan	53620	64	27	HT	39	160	2.2	1.8	137	4.8	Tri	T3	55	10.6	10.2	138	4.9	1.8		-	1.8
arjunan	31384	75	25	DM	55	180	2.8	2.2	138	5.2	Bi	T3	65	10.6	10.2	139	5.3	1.3		death due to M	exp
ramu	1174	60	27	DM	33	150	2.4	1.4	136	4.3	Bi	T3	45	11	10.2	135	4.4	1.5		-	1.3
abdul	48902	68	26	DM,HT	54	170	2.3	1.4	138	4.4	Tri	T2	45	10.8	10.2	137	4.6	1.4		-	1.4
selvam	48094	61	26		27	180	2.3	1.5	139	4.8	Bi	T3	55	10.9	9.5	136	4.8	1.6	Y	infection	1.5
mayakrisha	32832	73	23		50	150	1.8	1.6	141	4.7	Tri	T2	50	12	11.4	140	4.8	1.5		-	1.6
ganesan	55761	65	28		24	170	1.8	1.8	137	4.6	Bi	T2	40	12	10	141	4.8	1.4	Y	bleeding	1.8
devadurai	48091	63	23		50	180	1.6	1.6	138	4.6	Tri	T2	60	12	11.3	138	4.5	1.7		-	1.6
subramani	53340	60	26		35	160	2	1.6	136	4.3	Bi	T3	50	11.6	11.4	139	4.4	1.6		retn	1.6
gandhinath	34075	63	23		36	170	1.5	1.5	138	4.5	Bi	T2	50	10.8	10.5	140	4.6	1.8		-	1.5
surendran	57819	65	24		32	150	1.4	1.4	139	4.5	Bi	T2	50	11	10	138	4.7	1.4		-	1.4
nithyanand	38403	73	28	DM	55	200	2	1.1	141	4.7	Tri	T3	50	10.8	9.8	138	4.9	1.6		retn	1.1
palanivel	38253	70	23		53	150	2.3	1	138	4.6	Tri	T2	60	11.6	11.4	139	4.6	1.8		-	1
subbarayan	36457	75	25		59	160	2.3	1.1	136	4.4	Tri	T3	62	11.4	11	138	4.7	1.7		-	1.1
benedict	15688	60	25	HT	55	140	1.8	1.1	138	4.4	Tri	T2	60	10.8	10.3	137	4.6	1.7		-	1.1
kulandai	15695	65	26		42	130	1.7	1.2	140	4.6	Bi	T2	50	11	10	139	4.7	1.6			1.2
Usman	4085	61	25	HT	31	120	0.8	0.8	141	4.5	Bi	T3	60	11	10	138	4.8	0.8	Y	bleeding	0.9
santhanam	52431	70	26	HT	63	140	0.9	0.9	138	4.6	Tri	T2	55	12	11.8	139	4.8	0.9		-	0.8
sankar	56622	57	23	DM	23	136	1	1	136	4.6	Bi	T2	50	12	11.5	140	4.5	1		-	1
manickam	25771	74	27	DM	58	120	1.1	1.1	138	4.6	Tri	T3	70	12	11.4	140	4.6	1.1		-	1.1
palanisamy	58226	65	27		62	120	0.9	0.9	140	4.7	Tri	T3	90	11.6	11.4	134	4.8	0.9			0.9
moookapill	16203	73	21		36	180	0.8	0.8	141	4.7	Tri	T2	50	11.8	11.6	138	4.8	0.8	Y	bleeding	0.8
arumugam	17200	67	23		26	130	0.9	0.9	137	4.8	Bi	T2	50	11.7	11.4	137	4.8	0.9		-	0.9
balasubram	12193	62	26		50	140	1	1	138	4.5	Tri	T2	60	10.8	10.6	135	4.5	1		-	1
padmanabh	26921	65	24	HT,DM	50	130	0.9	0.9	136	4.8	Tri	T2	50	11	10.2	136	4.8	0.9		-	0.9
abdul shuk	29153	60	25		39	90	0.9	0.9	138	4.7	Tri	T2	50	10.8	10.2	138	4.5	1		-	1
thulasiram	37724	57	24		25	130	1	1	139	4.6	Bi	T3	50	10.9	9.5	139	4.9	1.1		-	1
kaniappan	38957	60	25	HT	28	140	1.1	1.1	141	4.7	Bi	T2	60	10.8	9.8	135	4.9	1.1		+	1.1
dhanaraj	36668	72	26	DM	55	170	0.7	0.7	138	4.6	Tri	T3	45	11.6	11.4	137	4.8	0.8		-	0.7
chellan	44494	63	23		30	90	0.8	0.8	138	4.7	Bi	T2	50	11.4	11	136	4.7	0.9		-	0.8
gnanasigan	48865	60	27		60	100	0.9	0.9	140	4.6	Tri	T2	70	11	10	138	4.8	0.9		-	0.8
gopi	40974	60	27		58	170	1	1	141	4.5	Tri	T2	50	10.7	10	139	4.9	1		retn	1
mohd ali	44494	54	21		36	130	1.1	1.1	137	4.5	Tri	T2	50	10.8	10.2	138	4.4	1		-	1.1
munusamy	48228	52	23	DM	26	150	0.8	0.8	138	4.5	Bi	T2	50	10.9	9.5	139	4.5	0.8		retn	0.8
pandian	46854	72	26		52	170	0.9	0.9	136	4.5	Tri	T3	50	10.8	9.8	141	4.6	0.9		-	0.9
ambikapath	40614	76	24		50	180	1	1	138	4.7	Tri	T2	60	11.6	11.4	140	4.8	1		-	1

S.creatinine 7 th day	Post op PVR	S.creatinine 6 weeks
1.8	20	1.8
2	12	2
1.9	15	1.9
1.9	35	1.9
1.7	45	1.7
exp	exp	exp
1.3	100	1.3
1.4	120	1.4
1.4	25	1.4
1.5	40	1.5
1.4	25	1.4
1.5	30	1.5
1.6	on catheter	1.6
1.3	25	1.3
1.2	30	1.2
1	on catheter	1
1	20	1
1	40	1
1	35	1
1.1	15	1.1
8	30	0.8
0.9	20	0.9
1	15	1
1	20	1
0.8	30	0.8
1	40	1
0.9	30	0.9
0.9	25	0.9
1	30	1
0.8	20	0.8
1	40	1
1	30	1
0.8	40	0.8
0.9	50	0.9
0.7	40	0.7
0.8	on cathete	0.8
1	20	1
0.8	on cathete	0.8
0.8	30	0.8
0.9	40	0.9

KEY TO MASTER CHART

Bi	-	Bilobar
Exp	-	Expired
He	-	Haemorrhage
Refr	-	Refractory renal failure
Retn	-	Retention
T2	-	Trabeculation grade II
T3	-	Trabeculation grade III
Tri	-	Trilobar
DM	-	Diabetes Mellitus
HTN	-	Hypertension